Abstract

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Atrial fibrillation (AF) in β-thalassemiapatients: role of DOACs for thromboembolic prophylaxis

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Background

Beta-thalassemia is a hereditary disease with worldwide distribution characterized by defective hemoglobin synthesis. Transfusion therapy and iron chelation have radically improved the prognosis of beta-thalassemia patients, however also allowing the development of new chronic, cardiac and non-cardiac complications (diabetes, dysthyroidism, autonomic disorders and many others), among which atrial fibrillation stands out. The prevalence of AF in patients with beta-thalassemia is dramatically higher than in the general population, ranging from 2 to 33%. Studies are lacking, and the little evidence available comes from a small number of observational studies. The pathophysiology underlying AF in thalassemia is not well understood; the responsible mechanisms are multiple and different from those recognized in the general population. Atrial iron overload appears to be the main "risk factor", although this arrhythmia can develop even before cardiac siderosis. The clinical presentation is early and mainly paroxysmal, with highly symptomatic patients. Furthermore, the numerous treatments available for AF (pharmacological and interventional), extensively studied in the general population, are largely unexplored in beta-thalassemia. All this requires a specific management of these patients; in particular, rhythm control should be preferred over rate control, and the most important antiarrhythmic therapy is represented by chelation drugs. Thromboembolic risk is also higher than in the general population and the choice of anticoagulant therapy must be considered early (well considering the serious consequences of a haemorrhage in this context), but the risk scores available for patients with AF are not validated in beta-thalassemia. The use of DOACs for thromboembolic prophylaxis in patients with β-thalassemia has not been systematically evaluated.

Methods

We enrolled patients with transfusion-dependent β-thalassemia, who where on treatments with DOACs for thromboembolic prophylaxis. Data on thromboembolic and bleeding events were collected.

Results

Eighteen patients were enrolled (10 male; 8 female – median age 62 years). The patients had a history of AF. The patients were treated whith dabigatran (8), apixaban (5); rivaroxaban (2); edoxaban (3). In the follow-up (mean follow-up duration: 28 months), no thromboembolic events were reported and only two patients had non-major bleeding (1 male had hematuria and 1 female had epistaxis). We observed only one case of major bleeding (male 64y) for non-fatal cerebral hemorrhage (right intra-axial fronto-basal hemorrhagic focus -dimensions max of 60x32 mm). This patient, suffering from HCC on HCV-related liver cirrhosis, had started treatment with levatinib 12 mg/die, (started three days before admission for major bleeding) a drug whose bleeding risks are known.

Discussion

Our data confirm the safety and efficacy of direct oral anticoagulants in patients affected by β-thalassemia.